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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/511,545	06/13/2005	Thomas Hesterkamp	P67818US1	6434

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EXAMINER	
LIU, SUE XU	
ART UNIT	**PAPER NUMBER**
1639	
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/511,545	HESTERKAMP ET AL.	
	Examiner	Art Unit	
	SUE LIU	1639	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *07 December 2007*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-20* is/are pending in the application.

4a) Of the above claim(s) *8-20* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-7* is/are rejected.

7)☒ Claim(s) *3* is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *10/18/04* is/are: a)☐ accepted or b)☒ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☒ All b)☐ Some * c)☐ None of:

1.☒ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *6/22/05*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Claim Status

1. Claims 1-20 are currently pending.

Claims 8-20 have been withdrawn.

Claims 1-7 are being examined in this application.

Election/Restrictions

2. Applicant's election of Group 1 (claims 1-7) in the reply filed on 12/17/07 is acknowledged. Because applicant did not distinctly and specifically point out the supposed errors in the restriction requirement, the election has been treated as an election without traverse (MPEP § 818.03(a)).

3. Claims 8-20 are withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to nonelected inventions, there being no allowable generic or linking claim. Election was made **without** traverse in the reply filed on 12/17/07.

4. Applicant's election without traverse of the following species:

A.) diagnosing;

in the reply filed on 12/17/2007 is acknowledged.

Priority

5. This application is filed under 35 U.S.C 371 of PCT/EP03/04058 (filed on 04/17/2003), which appears to claim priority to US provisional applications 60/373,375 (filed on 4/18/2002).

6. The instant application also claims priority to a foreign application EP 02008701.1 (filed on 4/18/02). Receipt is acknowledged of papers submitted under 35 U.S.C. 119(a)-(d), which papers have been placed of record in the file.

Information Disclosure Statement

7. The IDS filed on 6/22/05 has been considered. See the attached PTO 1449 form.

Drawings

8. The drawings/figures are objected to because tables and sequence listings included in the specification must not be duplicated in the drawings. (see Figure 6). See 37 C.F.R. §1.58(a) and §1.83. Applicants are advised that upon issuance of a patent, the complete text of the sequence listing submitted in compliance with 37 C.F.R.§§1.821-1.825 will be published as part of the patent. Applicants should amend the specification to delete any Figures which consist only of nucleic acid or protein sequences which have been submitted in their entirety in computer readable format (i.e. as SEQ ID NO:'s) and should further amend the specification accordingly to reflect the replacement of the Figure by the appropriate SEQ ID NO:.

Appropriate correction is required.

Specification

Sequence Rule Compliance

9. "In order to expedite the processing of applications, minor errors pertaining to compliance with the sequence rules may be handled with the first Office action." See MPEP 2427.01.

10. This application contains sequence disclosures that are encompassed by the definitions for nucleotide and/or amino acid sequences set forth in 37 CFR § 1.821(a)(1) and (a)(2). However, this application fails to comply with the requirements of 37 CFR §§ 1.821 through 1.825 for the reason(s) below:

The instant disclosure recites lists of sequences in the specification (e.g. see p. 31), which sequences are not identified by their corresponding SEQ ID Nos. The instant disclosure also recites lists of sequences in the drawings (see Figure 5), which sequences are not identified by their corresponding SEQ ID Nos in the "BRIEF DESCRIPTION OF THE FIGURES AND TABLES" of the instant specification.

In order to be fully responsive to the instant office action, applicants must fully comply with the Sequence Rule.

11. The specification has not been checked to the extent necessary to determine the presence of all possible minor errors. Applicant's cooperation is requested in correcting any errors of which applicant may become aware in the specification. MPEP 608.01.

Claim Objections

12. Claim 3 is objected to because of the following informalities: The term "tor" following the term "treatment" in the preamble of the claim should be "for." Appropriate correction is required.

Claim Rejections - 35 USC § 112

13. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Written Description Rejection

14. Claims 1-7 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The instant claims (claim 1) recite "A method of diagnosing or prognosticating a neurodegenerative disease in a subject, or determining whether a subject is at increased risk of developing said disease, comprising: determining a level and/or an activity of

(i) a transcription product of the ABCA 1 gene, and/or (ii) a translation product of the ABCA 1 gene, and/or (iii) a fragment, or derivative, or variant of said transcription or translation product,

in a sample from said subject and comparing said level and/or said activity to a reference value representing a known disease or health status, thereby diagnosing or prognosticating said neurodegenerative disease in said subject, or determining whether said subject is at increased risk of developing said neurodegenerative disease.

The instant claims (claims 2 and 3) are also drawn to methods of "monitoring progression" or "evaluating a treatment" of "a neurodegenerative disease"

To satisfy the written description requirement, applicants may convey reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession of the invention.

Applicants may show possession of an invention by disclosure of drawings or structural chemical formulas that are sufficiently detailed to show that applicant was in possession of the claimed invention as a whole. See, e.g., Vas-Cath, 935 F.2d at 1565, 19 USPQ2d at 1118.

The written description requirement of 35 U.SC. 112 exists independently of enablement requirement, and the requirement applies whether or not the case involves questions of priority. The requirement applies to all inventions, including chemical inventions, and because the fact that the patent is directed to method entailing use of compound, rather than to compound per se, does not remove patentee's obligation to provide a description of the compound sufficient to distinguish infringing methods from non-infringing methods. See Univ. of Rochester v. G.D. Searle & Co., 358 F.3d 916, 920-23, 69 USPQ 2d 1886, 1890-93 (Fed. Cir. 2004).

With regard to the description requirement, applicants' attention is invited to consider the decision of the Court of Appeals for the Federal Circuit, which holds that a "written description of an invention involving a chemical genus, like a description of a chemical species, 'requires a precise definition, such as by structure, formula [or] chemical name,' of the claimed subject matter sufficient to distinguish it form other materials." University of California v. Eli Lilly and Co., 43 USPQ2d 1398, 1405 (1997), quoting Fiers v. Revel, 25 USPQ2d 1601, 1606 (Fed. Cir. 1993) (bracketed material in original) [The claims at issue in University of California v. Eli Lilly defined the invention by function of the claimed DNA (encoding insulin)].

The written description requirement for a claimed genus may be satisfied through sufficient description of a representative number of species or by actual reduction to practice, reduction to drawings, or by disclosure of relevant, identifying characteristics, i.e., structure or other physical an/or chemical properties, by functional characteristics coupled with a known or disclosed correlation between function and structure, or by a combination of such identifying characteristics, sufficient to show the applicant was in possession of the claimed genus. See Eli Lilly, 119 F. 3d at 1568, 43 USPQ2d at 1406.

The instant claim 1 is construed to recite a method for "diagnosing or prognosticating a neurodegenerative disease" by determining gene expression level and/or activity of the ABCA 1 gene (or the ATP-binding cassette transporter A1). Similarly, the instant claims 2 and 3 are also broadly drawn to methods of "monitoring progression" or "evaluating a treatment" for "a neurodegenerative disease". Claims 1-3 as written are broadly drawn to a genus of methods for a

genus of "diseases" (including Alzheimer's disease) using a genus of "fragments/variants" of the ABCA 1 gene and/or protein.

Neither the instant specification nor the claims have demonstrated common structure and/or function for the claimed genus of methods of "diagnosing," "prognosticating," "monitoring progression," and "evaluating a treatment" using ABCA 1 gene/protein or fragments/variants thereof. In addition, no representative numbers of species for each claimed genus is provided to show possession of the claimed genuses.

To provide evidence of possession of a claimed genus, the specification must provide sufficient distinguishing identifying characteristics of the genus. The factors to be considered include disclosure of complete or partial structure, physical and/or chemical properties, functional characteristics, structure/function correlation, methods of making the claimed product, or any combination thereof. (see MPEP 2163 II).

In this case, the instant specification is general and prophetic in nature. The instant specification does NOT provide any example for the methods of diagnosing, prognosticating, monitoring progression, and evaluating a treatment using ABCA 1 gene/protein or fragments/variants thereof. The instant specification also does not provide representative number of species or a common structure/function for the claimed genus of "fragments/derivatives/variants" of the ABCA1 gene/protein.

The only examples provided in the instant disclosure are methods of measuring mRNA level of the ABCA1 gene from post-mortem human brain tissues (spec. pp.30+). No example of using the expression and/or activity level of the ABCA 1 gene as a tool for diagnosing for a particular neurodegenerative disease was provided. In the only example, Example I (spec.,

pp.30+), the mRNA level of the ABCA1 gene is compared between two different areas of the brain within the same individual. It is not clear from the instant disclosure that any differential expression was observed (i.e. no clear data). There is also no data to show that ABCA1 gene is differentially expressed between a normal and a person with a neurodegenerative disease such as Alzheimer's Disease (AD). Furthermore, the instant specification has not demonstrated the possession of a method using the expression/activity levels of the ABCA1 gene/protein to diagnose/prognosticate for a neurodegenerative disease. It is not clear if the observed differences (if any) can be conclusively correlated to a positive diagnosis for Alzheimer's Disease. More importantly, applicants have not shown that a determination of the ABCA1 gene/protein level/activity will conclusively lead to a successful diagnosis/prognosis (or other determination) of any neurodegenerative disease including AD.

In addition, the instant specification also does not provide core structure that is required for the claimed genus of "fragments", "variants" or "derivatives" of the ABCA1 gene/protein. The terms "fragments", "variants" or "derivatives" can be construed broadly and reasonably to mean a gene or a protein having one or more nucleic (or amino) acid substitutes, deletions, insertions and/or additions made to the ABCA1 gene/protein. For example, the instant claims would encompass using a fragment of 2 nucleotides/amino acids of the ABCA1 gene/protein for the claimed method. The instant specification does not provide how these various fragments/variants can be used specifically to monitor or diagnose diseases. Further, the specification and claim do not indicate what distinguishing attributes are shared by the members of the genus (of fragments/variants). The instant specification and claims do not place any limit on the number of nucleic-acid/amino-acid substitutions, deletions, insertions and/or additions

that may be made to the wildtype ABCA1 gene/protein. Thus, the scope of the instant claims includes numerous structural variants, and the genus is highly variable because a significant number of structural differences between genus members is permitted. Although the instant specification discloses the ABCA 1 protein have an amino acid sequence of SEQ ID NO:1, the specification and claims do not provide any guidance as to what specific changes should be made to the said sequence. Structural features that could distinguish compounds in the genus from others in the protein class are missing from the disclosure. No common structural attributes identify the members of the genus. The general knowledge and level of skill in the art do not supplement the omitted description because specific, not general, guidance is what is needed. Since the disclosure fails to describe the common attributes or characteristics that identify members of the genus, and because the genus is highly variant, the wildtype ABCA 1 gene/protein alone is insufficient to describe the genus. One of skill in the art would reasonably conclude that the disclosure fails to provide a representative number of species to describe the genus. Thus, applicant was not in possession of the claimed genus.

Furthermore, it is not known in the art that an observed differential ABCA1 gene expression pattern or a change in the ABCA1 protein activity will lead to various neurodegenerative diseases; it is also not known ABCA1 gene/protein level/activity specifically correlate with neurodegenerative diseases.

Currently, there is no effective or even reliable diagnostic method for neurodegenerative diseases such as Alzheimer's disease. For examples, **Patel**, (Journal of Geriatric Psychiatry and Neurology, Vol. 8, 81-95; 1995) discloses that Alzheimer's disease is known to be difficult to treat and that there is neither a clear understanding of the origin and pathophysiology of AD

(Alzheimer's disease) nor an animal model of the illness (see Patel, page 81). The reference states that the search for an effective cognition-enhancing therapy for AD has so far proved to be elusive (see Patel, page 90). Similarly, **Nagasaka** et al (PNAS. vol. 102(41): 14854-14859; 2005) also state the current diagnostic tools have poor sensitivity for diagnosing the onset of AD, and it may take several years from onset of cognitive decline to diagnosis (p.14854, left col.). Furthermore, **Jacobsen** et al, (Neuro Rx: The Journal of the American Society for Experimental NeuroTherapeutics. Vol. 2: 612-626; 10/2005; published after the instant filing date), state that "antemortem clinical diagnosis of AD is difficult" and "unqualified diagnosis of AD can still only be made neuropathologically postmortem" (p.612, col.1-2, bridging). That is diagnosis of AD is difficult and highly unpredictable.

It is not known the expression profile or activity profile of one particular gene (e.g. ABCA1) or its fragments/variants can conclusively lead to a positive diagnosis. In fact, a predictable relationship between ABCA1 gene/protein and AD development has not been conclusive demonstrated in the art. For example, **Hirsch-Reinshagen** et al (Journal of Biological Chemistry. Vol. 280(52): 43243-43243; 12/30/2005) state a lack of ABCA1 in mouse models did result in reduction in soluble apoE levels, however, the reduction in apoE levels did not decrease "amyloid burden" (a postulated symptom in AD patients) (p.43244, col.2, para 3). The reference also states that certain single nucleotide polymorphism in ABCA1 has been suggested to associated with AD, however, "these findings were not replicated in a large case-control study" and "further investigations will therefore be required to determine whether inactivation of ABCA1 affects AD in humans" (p.43254, col.2, para 2). Thus, the relationship between ABCA1

gene/protein and AD in human is highly unpredictable and using ABCA1 gene/protein level/activity to diagnose neurodegenerative diseases such as AD is also highly unpredictable.

In addition, the underlying mechanism for AD is still not clearly understood. It is not known what specific gene(s) and/or molecule cause the disease. For example, the specific relationship between certain substances such as amyloid β and the physiological consequences of AD is still not clearly understood (see **Kar** et al, J. Psychiatry Neurosci. Vol. 29(6): 427-441; 2004).

Thus, the state of the art (even after the effective filing date of the instant application) demonstrates diagnosis of AD are highly unpredictable.

Additionally, the in vitro data provided given the unpredictability of the art would not be viewed as correlative to human applications. In vivo application necessarily involves unpredictability with respect to physiological activity of an asserted process in humans. See discussion in Ex parte Kranz, 19 USPQ2d 1216,1218-1219 (6/90).

Therefore, applicants are not in possession of the entire claimed methods diagnosing or prognosticating a neurodegenerative disease" by determining gene expression level and/or activity of the ABCA 1 gene/protein or fragments/variants/derivatives thereof. Applicant's claimed scope represents only an invitation to experiment regarding possible methods that might be used to diagnose/prognosticate various neurodegenerative diseases.

Enablement Rejection

15. Claims 1-7 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in

the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

Factors to be considered in determining whether a disclosure meets the enablement requirement of 35 U.S.C. §112, first paragraph, have been described *In re Wands*, 8 USPQ2d 1400(1988). They are:

1. The breadth of the claims;
2. The nature of the invention;
3. The state of the prior art;
4. The predictability or lack thereof in the art
5. The level of skill in the art;
6. The amount of direction or guidance present;
7. The presence or absence of working examples;
8. The quantity of experimentation needed.

The breadth of the claims / The nature of the invention

The instant claim 1 is construed to recite a method for "diagnosing or prognosticating a neurodegenerative disease" by determining gene expression level and/or activity of the ABCA 1 gene (or the ATP-binding cassette transporter A1). Similarly, the instant claims 2 and 3 are also broadly drawn to methods of "monitoring progression" or "evaluating a treatment" for "a neurodegenerative disease". Claims 1-3 as written are broadly drawn to a genus of methods for a genus of "diseases" (including Alzheimer's disease) using a genus of "fragments/variants" of the ABCA 1 gene and/or protein.

The state of the prior art/ The predictability or lack thereof in the art

It is not known in the art that an observed differential ABCA1 gene expression pattern or a change in the ABCA1 protein activity will lead to various neurodegenerative diseases; it is also not known ABCA1 gene/protein level/activity specifically correlate with neurodegenerative diseases.

Currently, there is no effective or even reliable diagnostic method for neurodegenerative diseases such as Alzheimer's disease. For examples, **Patel**, (Journal of Geriatric Psychiatry and Neurology, Vol. 8, 81-95; 1995) discloses that Alzheimer's disease is known to be difficult to treat and that there is neither a clear understanding of the origin and pathophysiology of AD (Alzheimer's disease) nor an animal model of the illness (see Patel, page 81). The reference states that the search for an effective cognition-enhancing therapy for AD has so far proved to be elusive (see Patel, page 90).

Similarly, **Nagasaka** et al (PNAS. vol. 102(41): 14854-14859; 2005) also state the current diagnostic tools have poor sensitivity for diagnosing the onset of AD, and it may take several years from onset of cognitive decline to diagnosis (p.14854, left col.).

Furthermore, **Jacobsen** et al, (Neuro Rx: The Journal of the American Society for Experimental NeuroTherapeutics. Vol. 2: 612-626; 10/2005; published after the instant filing date), state that "antemortem clinical diagnosis of AD is difficult" and "unqualified diagnosis of AD can still only be made neuropathologically postmortem" (p.612, col.1-2, bridging). That is diagnosis of AD is difficult and highly unpredictable.

It is also not known the expression profile or activity profile of one particular gene (e.g. ABCA1) or its fragments/variants can conclusively lead to a positive diagnosis. In fact, a

predictable relationship between ABCA1 gene/protein and AD development has not been conclusive demonstrated in the art. For example, **Hirsch-Reinshagen** et al (Journal of Biological Chemistry. Vol. 280(52): 43243-43243; 12/30/2005) state a lack of ABCA1 in mouse models did result in reduction in soluble apoE levels, however, the reduction in apoE levels did not decrease "amyloid burden" (a postulated symptom in AD patients) (p.43244, col.2, para 3). The reference also states that certain single nucleotide polymorphism in ABCA1 has been suggested to associated with AD, however, "these findings were not replicated in a large case-control study" and "further investigations will therefore be required to determine whether inactivation of ABCA1 affects AD in humans" (p.43254, col.2, para 2). Thus, the relationship between ABCA1 gene/protein and AD in human is highly unpredictable and using ABCA1 gene/protein level/activity to diagnose neurodegenerative diseases such as AD is also highly unpredictable.

In addition, the underlying mechanism for AD is still not clearly understood. It is not known what specific gene(s) and/or molecule cause the disease. For example, the specific relationship between certain substances such as amyloid β and the physiological consequences of AD is still not clearly understood (see **Kar** et al, J. Psychiatry Neurosci. Vol. 29(6): 427-441; 2004).

Thus, the state of the art (even after the effective filing date of the instant application) demonstrates diagnosis of AD are highly unpredictable.

Additionally, the in vitro data provided given the unpredictability of the art would not be viewed as correlative to human applications. In vivo application necessarily involves unpredictability with respect to physiological activity of an asserted process in humans. See discussion in Ex parte Kranz, 19 USPQ2d 1216,1218-1219 (6/90).

The level of one of ordinary skill

The level of skill would be high, most likely at the Ph.D. level.

The amount of direction or guidance present / The presence or absence of working examples

In this case, the instant specification is general and prophetic in nature. The instant specification does NOT provide any example for the methods of diagnosing, prognosticating, monitoring progression, and evaluating a treatment using ABCA 1 gene/protein or fragments/variants thereof. The instant specification also does not provide representative number of species or a common structure/function for the claimed genus of "fragments/derivatives/variants" of the ABCA1 gene/protein.

The only examples provided in the instant disclosure are methods of measuring mRNA level of the ABCA1 gene from post-mortem human brain tissues (spec. pp.30+). No example of using the expression and/or activity level of the ABCA 1 gene as a tool for diagnosing for a particular neurodegenerative disease was provided. In the only example, Example I (spec., pp.30+), the mRNA level of the ABCA1 gene is compared between two different areas of the brain within the same individual. It is not clear from the instant disclosure that any differential expression was observed (i.e. no clear data). There is also no data to show that ABCA1 gene is differentially expressed between a normal and a person with a neurodegenerative disease such as Alzheimer's Disease (AD). Furthermore, the instant specification has not demonstrated a method using the expression/activity levels of the ABCA1 gene/protein to diagnose/prognosticate for a neurodegenerative disease in any subject. It is not clear if the observed differences (if any) can

be conclusively correlated to a positive diagnosis for Alzheimer's Disease. More importantly, applicants have not shown that a determination of the ABCA1 gene/protein level/activity will conclusively lead to a successful diagnosis/prognosis (or other determination) of any neurodegenerative disease including AD.

The quantity of experimentation needed

Due to the unpredictabilities of using ABCA1 gene/protein (or fragments/variants/derivatives thereof) level/activity to diagnose/prognosticate various neurodegenerative diseases including AD, undue experimentation would be required. The state of art does not provide reliable or predictable methods of correlating ABCA1 gene/protein (or fragments/variants/derivatives thereof) to AD or other neurodegenerative diseases. In addition, neither the instant specification nor the art has demonstrated that any "fragments", and "variants" of ABCA1 gene/protein can be used for the claimed assay. Because the instant specification only provides guidance for measuring mRNA levels of ABCA1 gene in postmortem brains, undue experimentation would be required to practice claimed method of diagnosis/prognosis.

Conclusion

Therefore based on the evidences as a whole regarding each of the above factors (e.g. factors 1-8), the specification, at the time the application was filed, does not satisfy the enablement requirement for the instant claimed method.

Conclusion

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Sue Liu whose telephone number is 571-272-5539. The examiner can normally be reached on M-F 9am-3pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Doug Schultz can be reached at 571-272-0763. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

/Sue Liu/
Patent Examiner, AU 1639
2/25/07